10/075074
File 5:Biosis Previews(R) 1969-2004/Sep W2
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Set	Items	Description
S1	18225	(POTASSIUM()CHANNEL)
S2	403	(BIND(5W) SUBUNIT)
S3	5	S1 AND S2
S4	213	S1 AND TOXINS
S5	0	S1 AND (CYTOPLASM?3W TOXINS)
S6	0	S1 AND (CYTOPLASM? (3W) TOXINS)
S7	0	S1 AND (ASSOCIATED()PROTIENS)
S8	17	S1 AND (ASSOCIATED()PROTEINS)
S9	129	AU='BROWN ARTHUR' OR AU='BROWN ARTHUR M'
S10	50	AU='WIBLE BARBARA' OR AU='WIBLE BARBARA A'
S11	172	AU='YANG QING'
S12	44	S1 AND S9
S13	29	S1 AND S10
S14	7	S1 AND S11
S15	6	S12 AND S14

? t s3/7/1-5

3/7/1
DIALOG(R)File 5:Biosis Previews(R)

0014861755 BIOSIS NO.: 200400231464
Interaction of ATP sensor, cAMP sensor, Ca2+ sensor, and voltage-dependent Ca2+ channel in insulin granule exocytosis.
AUTHOR: Shibasaki Tadao; Sunaga Yasuhiro; Fujimoto Kei; Kashima Yasushige; Seino Susumu (Reprint)
AUTHOR ADDRESS: Division of Cellular and Molecular Medicine, Kobe University Graduate School of Medicine, Kobe, 650-0017, Japan**Japan
AUTHOR E-MAIL ADDRESS: seino@med.kobe.u.ac.jp
JOURNAL: Journal of Biological Chemistry 279 (9): p7956-7961 February 27, 2004 2004
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: ATP, cAMP, and Ca2+ are the major signals in the regulation of insulin granule exocytosis in pancreatic beta cells. The sensors and regulators of these signals have been characterized individually. The ATP-sensitive K+ channel, acting as the ATP sensor, couples cell metabolism to membrane potential. cAMP-GEFII, acting as a cAMP sensor, mediates cAMP-dependent, protein kinase A-independent exocytosis, which requires interaction with both Piccolo as a Ca2+ sensor and Rim2 as a Rab3 effector. L-type voltage-dependent Ca2+ channels (VDCCs) regulate Ca2+ influx. In the present study, we demonstrate interactions of these molecules. Sulfonylurea receptor 1, a subunit of ATP-sensitive K+ channels, interacts specifically with cAMP-GEFII through nucleotide-binding fold 1, and the interaction is decreased by a high concentration of cAMP. Localization of cAMP-GEFII overlaps with that of Rim2 in plasma membrane of insulin-secreting MIN6 cells. Localization of Rab3 coincides with that of Rim2. Rim2 mutant lacking the Rab3 binding region, when overexpressed in MIN6 cells, is localized exclusively in cytoplasm, and impairs cAMP-dependent exocytosis in MIN6 cells. In addition, Rim2 and Piccolo %%%bind%%% directly to the alpha11.2-%%%subunit%%% of VDCC. These results indicate that ATP sensor, cAMP sensor, Ca2+ sensor, and VDCC interact with each other, which further suggests that ATP, cAMP, and Ca2+ signals in insulin granule exocytosis are integrated in a specialized domain of pancreatic beta cells to facilitate stimulus-secretion coupling.

3/7/2
DIALOG(R)File 5:Biosis Previews(R)

0014465649 BIOSIS NO.: 200300434368
Pyridine nucleotide regulation of the KATP channel Kir6.2/SUR1 expressed in Xenopus oocytes.
AUTHOR: Dabrowski Michael; Trapp Stefan; Ashcroft Frances M (Reprint)
AUTHOR ADDRESS: University Laboratory of Physiology, Parks Road, Oxford, OX1 3PT, UK**UK
AUTHOR E-MAIL ADDRESS: frances.ashcroft@physiol.ox.ac.uk
JOURNAL: Journal of Physiology (Cambridge) 550 (2): p357-363 15 July, 2003 2003
MEDIUM: print
ISSN: 0022-3751 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The pancreatic beta-cell type of ATP-sensitive potassium (KATP) channel (Kir6.2/SUR1) is inhibited by intracellular ATP and ADP, which %%%bind%%% to the Kir6.2 %%%subunit%%%, and is activated by Mg-nucleotide interaction with the regulatory sulphonylurea receptor subunits (SUR1). The nicotinamide adenine dinudeotides NAD and NADP consist of an ADP molecule with a ribose group and a nicotinamide moiety attached to the terminal phosphate. Both these molecules block native KATP channels in pancreatic beta-cells at concentrations above 500 muM, and activate them at lower concentrations. We therefore investigated whether NAD and NADP interact with both Kir6.2 and SUR1 subunits of the KATP channel by comparing the potency of these agents on recombinant Kir6.2DELTAC and Kir6.2/SUR1 channels expressed in Xenopus oocytes. Our results show that, at physiological concentrations, NAD and NADP interact with the nucleotide inhibitory site of Kir6.2 to inhibit Kir6.2/SUR1 currents. They may therefore contribute to the resting level of channel inhibition in the intact cell. Importantly, our data also reveal that this interaction is dependent on the presence of SUR1, which may act by increasing the width of the nucleotide-binding pocket of Kir6.2.

3/7/3
DIALOG(R)File 5:Biosis Previews(R)

0013748339 BIOSIS NO.: 200200341850
Modulation of Drosophila slowpoke calcium-dependent %%%potassium%%% %%%channel%%% activity by bound protein kinase A catalytic subunit
AUTHOR: Zhou Yi; Wang Jing; Wen Hua; Kucherovsky Olga; Levitan Irwin B (Reprint)
AUTHOR ADDRESS: Department of Neuroscience, University of Pennsylvania School of Medicine, Philadelphia, PA, 19104, USA**USA
JOURNAL: Journal of Neuroscience 22 (10): p3855-3863 May 15, 2002 2002
MEDIUM: print
ISSN: 0270-6474
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Drosophila Slowpoke (dSlo) calcium-dependent potassium channels %%%bind%%% directly to the catalytic %%%subunit%%% of cAMP-dependent protein kinase (PKAc). We demonstrate here that coexpression of PKAc with dSlo in mammalian cells results in a dramatic decrease of dSlo channel activity. This modulation requires catalytically active PKAc but is not mediated by phosphorylation of S942, the only PKA consensus site in the dSlo C-terminal domain. dSlo binds to free PKAc but not to the PKA holoenzyme that includes regulatory subunits and is native. Activators of endogenous PKA that stimulate dSlo phosphorylation, but do not produce detectable PKAc binding to dSlo, do not modulate channel function. Furthermore, the catalytically inactive PKAc mutant does bind to dSlo but does not modulate channel activity. These results are consistent with the hypothesis that both binding of active PKAc to dSlo and phosphorylation of dSlo or some other protein are necessary for channel modulation.

3/7/4
DIALOG(R)File 5:Biosis Previews(R)

0012920642 BIOSIS NO.: 200100092481
Modulation of dSlo channel activity by associated protein kinase A catalytic subunit
AUTHOR: Zhou Y (Reprint); Levitan I B
AUTHOR ADDRESS: University of Pennsylvania, Philadelphia, PA, USA**USA
JOURNAL: Society for Neuroscience Abstracts 26 (1-2): pAbstract No.-525.12 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000; 20001104
SPONSOR: Society for Neuroscience
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have shown previously that the cloned Drosophila Slowpoke calcium-dependent %%%potassium%%% %%%channel%%% (dSlo) can %%%bind%%% directly to the catalytic %%%subunit%%% of protein kinase A (PKAc). To study the effect of the bound kinase on dSlo channel activity, we have measured dSlo current with the whole-cell voltage-clamp configuration in a heterologous expression system. We found that co-expression of PKAc results in a decrease in dSlo channel activity. This is evidenced by a reduction of voltage sensitivity and a slowing of activation kinetics of dSlo currents in the presence of co-expressed PKAc. When dSlo is co-expressed with PKAc, the average peak current is lower than that in control, even though the amount of expressed dSlo protein is substantially increased. However, this down-regulation of dSlo channel activity is not mediated by the phosphorylation of the PKAc consensus site at serine 942 of dSlo (S942), because the activity of a mutant dSlo channel (S942A) is also down-regulated by co-expressed PKAc. These results provide evidence that dSlo channel activity can be influenced by a closely associated protein kinase. The precise molecular mechanism of this modulation remains to be determined.

3/7/5
DIALOG(R)File 5:Biosis Previews(R)

0008975628 BIOSIS NO.: 199396140044
Identification of 1,4-dihydropyridine binding domains within the primary structure of the alpha-1 subunit of the skeletal muscle L-type calcium channel
AUTHOR: Kalasz Huba; Watanabe Toshiro; Yabana Hideo; Itagaki Kiyoshi; Naito Kazuaki; Nakayama Hitoshi; Schwartz Arnold; Vaghy Pal L (Reprint)
AUTHOR ADDRESS: Dep. Med. Biochem., Ohio State Univ., 466 Hamilton Hall, 1645 Neil Avenue, Columbus, OH 43210-1218, USA**USA
JOURNAL: FEBS (Federation of European Biochemical Societies) Letters 331 (1-2): p177-181 1993
ISSN: 0014-5793
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Calcium channel blockers are drugs that %%%bind%%% to the alpha-1 %%%subunit%%% of L-type calcium channels and selectively inhibit ion movements through these channels. Determination of the mechanism of channel blockade requires localization of drug-binding sites within the primary structure of the receptor. In this study the 1,4-dihydropyridine-binding site of the membrane bound receptor has been identified. The covalently labeled receptor was purified and digested with trypsin. The labeled peptide fragments were immunoprecipitated with sequence-directed antibodies. The data indicate the existence of at least three distinct dihydropyridine-binding domains within the primary structure of the alpha-1, subunit.

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8/7/1
DIALOG(R)File 5:Biosis Previews(R)

0014936721 BIOSIS NO.: 200400307478
Protein trafficking and anchoring complexes revealed by proteomic analysis of inward rectifier %%%potassium%%% %%%channel%%% (Kir2.x)-%%%associated%%% %%%proteins%%%
AUTHOR: Leonoudakis Dmitri; Conti Lisa R; Anderson Scott; Radeke Carolyn M; McGuire Leah M M; Adams Marvin E; Froehner Stanley C; Yates John R; Vandenberg Carol A (Reprint)
AUTHOR ADDRESS: Dept Mol Cellular and Dev Biol, Univ Calif Santa Barbara, Santa Barbara, CA, 93106, USA**USA
AUTHOR E-MAIL ADDRESS: vandenbe@lifesci.ucsb.edu
JOURNAL: Journal of Biological Chemistry 279 (21): p22331-22346 May 21, 2004 2004
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inward rectifier potassium (Kir) channels play important roles in the maintenance and control of cell excitability. Both intracellular trafficking and modulation of Kir channel activity are regulated by protein-protein interactions. We adopted a proteomics approach to identify proteins associated with Kir2 channels via the channel C-terminal PDZ binding motif. Detergent-solubilized rat brain and heart extracts were subjected to affinity chromatography using a Kir2.2 C-terminal matrix to purify channel-interacting proteins. Proteins were identified with multidimensional high pressure liquid chromatography coupled with electrospray ionization tandem mass spectrometry, N-terminal microsequencing, and immunoblotting with specific antibodies. We identified eight members of the MAGUK family of proteins (SAP97, PSD-95, Chapsyn-110, SAP102, CASK, Dlg2, Dlg3, and Pals2), two isoforms of Veli (Veli-1 and Veli-3), Mint1, and actin-binding LIM protein (abLIM) as Kir2.2-associated brain proteins. From heart extract purifications, SAP97, CASK, Veli-3, and Mint1 also were found to associate with Kir2 channels. Furthermore, we demonstrate for the first time that components of the dystrophin-associated protein complex, including alpha1-, beta1-, and beta2-syntrophin, dystrophin, and dystrobrevin, interact with Kir2 channels, as demonstrated by immunoaffinity purification and affinity chromatography from skeletal and cardiac muscle and brain. Affinity pull-down experiments revealed that Kir2.1, Kir2.2, Kir2.3, and Kir4.1 all bind to scaffolding proteins but with different affinities for the dystrophin-associated protein complex and SAP97, CASK, and Veli. Immunofluorescent localization studies demonstrated that Kir2.2 co-localizes with syntrophin, dystrophin, and dystrobrevin at skeletal muscle neuromuscular junctions. These results suggest that Kir2 channels associate with protein complexes that may be important to target and traffic channels to specific subcellular locations, as well as anchor and stabilize channels in the plasma membrane.

8/7/2
DIALOG(R)File 5:Biosis Previews(R)

0014835879 BIOSIS NO.: 200400203512
Dystroglycan and the inwardly rectifying %%%potassium%%% %%%channel%%%, Kir4.1, CO - aggregate in glial cells.
AUTHOR: Guadagno E (Reprint); Doucet G; Moukhles H (Reprint)
AUTHOR ADDRESS: Sci. Biologiques, Universite de Montreal, Outremont, PQ, Canada**Canada
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner 2003 pAbstract No. 674.15 2003 2003
MEDIUM: e-file
CONFERENCE/MEETING: 33rd Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 08-12, 2003; 20031108
SPONSOR: Society of Neuroscience

DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Dystroglycan (DG) and %%%associated%%% %%%proteins%%% form a complex that links the extracellular matrix to the actin cytoskeleton of muscle fibers and that is involved in aggregating acetylcholine receptors at the neuromuscular junction. This complex is expressed in regions of the central nervous system where it is localized both to neuronal and glial cells. DG and Kir4.1 channels are concentrated at the interface of astroglia and small blood vessels, where these channels are involved in clearing the excess of potassium released after neuronal excitation. This raises the possibility that DG may be involved in targeting Kir4.1 channels to specific domains of astroglia. To address this question, we used mixed hippocampal cultures to investigate the distribution and localization of DG, syntrophin, dystrobrevin, Kir4.1 channels as well as aquaporin-4 water channels. These proteins exhibit a similar distribution to large aggregates in astrocytes cultured on laminin. Both DG and syntrophin colocalize with Kir4.1 channels in aggregates in astrocytes. Quantitative studies show that the density of Kir4.1 channel aggregates increases by 75% in astrocytes of hippocampus cultured on laminin compared with poly-D-lysine. Similarly, we found a significant increase in the number of DG and aquaporin-4 channel aggregates. Interestingly, the laminin-induced aggregates labeled for DG and Kir4.1 channels are also enriched for phosphotyrosine. These findings suggest a role for laminin and dystroglycan-containing complex(es) in targeting and aggregating Kir4.1 channels in astrocytic cells.

8/7/3
DIALOG(R)File 5:Biosis Previews(R)

0014620388 BIOSIS NO.: 200300571065
EXPRESSION AND CLUSTERED DISTRIBUTION OF THE %%%POTASSIUM%%% %%%CHANNEL%%% KIR4.1 AND AQUAPORIN 4 IN THE RETINA OF DYSTROPHIN PROTEIN DP71 NULL - MICE
AUTHOR: Rendon A (Reprint); Fort P (Reprint); Sarig R; Yaffe D; Nudel U; Pannicke T; Reichenbach A; Sahel J (Reprint); Dalloz C (Reprint)
AUTHOR ADDRESS: Lab. Physio Cell Molec Retine, INSERM-EMI 99-18, Paris, France**France
JOURNAL: ARVO Annual Meeting Abstract Search and Program Planner 2003 p Abstract No. 4581 2003 2003
MEDIUM: cd-rom
CONFERENCE/MEETING: Annual Meeting of the Association for Research in Vision and Ophthalmology Fort Lauderdale, FL, USA May 04-08, 2003; 20030504
SPONSOR: Association for Research in Vision and Ophthalmology
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose: To elucidate the role(s) of dystrophin protein Dp71 in retina we investigated in Dp71 null-mice the effect on dystrophin-%%%associated%%% %%%proteins%%% (DAPs) and on the expression and localization of the two membrane channel proteins, the inwardly rectifying %%%potassium%%% %%%channel%%% Kir4.1 and the aquaporin 4 (AQP4), which are responsible respectively for the control of potassium and water fluxes in Muller cells. Both channels may form clustered complexes with Dp71. Methods: The expression and localization of dystrophins, DAPs, Kir4.1 and AQP4 were determined by Western-blot and immunocytochemistry methods in retinal sections of wild and Dp71-null mice strains with specific antibodies. In double labeling experiments sections were incubated successively with both types of antibodies. All fluorescence specimens were viewed by using a confocal microscope Results: Analysis of dystrophins and DAPs showed that Dp71 was localized around blood vessels, at the inner limiting membrane (ILM) and that Dp71 deficiency results in decrease level of beta-dystroglycan localized in the ILM, without an apparent effect on the other DAPs. Dp71 deficiency was also associated with an impaired clustering of Kir4.1 and AQP4 around blood vessels and at the ILM. By Western-blot we remarked that the

absence of Dp71 induced the down-regulation of AQP4 without an effect on the Kir4.1 protein level Conclusions: Our results showed that Dp71 is involved in the stabilization of Kir4.1 potassium channels and AQP4, water channels at specialized membrane areas of Muller cells.

8/7/4
DIALOG(R)File 5:Biosis Previews(R)

0014397120 BIOSIS NO.: 200300355839
Targeted inactivation of dystrophin gene product Dp71: Phenotypic impact in mouse retina.
AUTHOR: Dalloz Cecile; Sarig Rachel; Fort Patrice; Yaffe David; Bordais Agnes; Pannicke Thomas; Grosche Jens; Mornet Dominique; Reichenbach Andreas; Sahel Jose; Nudel Uri; Rendon Alvaro (Reprint)
AUTHOR ADDRESS: Laboratoire de Physiopathologie Cellulaire et Moleculaire de la Retine, INSERM U-592, Hopital Saint-Antoine, 184 Rue du Faubourg Saint-Antoine, Batiment Kourilsky, 6eme Etage, 75571, Paris Cedex 12, France**France
AUTHOR E-MAIL ADDRESS: rendon@st-antoine.inserm.fr
JOURNAL: Human Molecular Genetics 12 (13): p1543-1554 1 July, 2003 2003
MEDIUM: print
ISSN: 0964-6906 (ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The abnormal retinal neurotransmission observed in Duchenne muscular dystrophy (DMD) patients and in some genotypes of mice lacking dystrophin has been attributed to altered expression of short products of the dystrophin gene. We have investigated the potential role of Dp71, the most abundant C-terminal dystrophin gene product, in retinal electrophysiology. Comparison of the scotopic electroretinograms (ERG) between Dp71-null mice and wild-type (wt) littermates revealed a normal ERG in Dp71-null mice with no significant changes of the b-wave amplitude and kinetics. Analysis of DMD gene products, utrophin and dystrophin-%%%associated%%% %%%proteins%%% (DAPs), showed that Dp71 and utrophin were localized around the blood vessels, in the ganglion cell layer (GCL), and the inner limiting membrane (ILM). Dp71 deficiency was accompanied by an increased level of utrophin and decreased level of beta-dystroglycan localized in the ILM, without any apparent effect on the other DAPs. Dp71 deficiency was also associated with an impaired clustering of two Muller glial cell proteins-the inwardly rectifying %%%potassium%%% %%%channel%%% Kir4.1 and the water pore aquaporin 4 (AQP4). Immunostaining of both proteins decreased around blood vessels and in the ILM of Dp71-null mice, suggesting that Dp71 plays a role in the clustering and/or stabilization of the two proteins. AQP4 and Kir4.1 may also be involved in the regulation of the ischemic process. We found that a transient ischemia resulted in a greater damage in the GCL of mice lacking Dp71 than in wt mice. This finding points at a crucial role played by Dp71 in retinal function.

8/7/5
DIALOG(R)File 5:Biosis Previews(R)

0014356854 BIOSIS NO.: 200300315573
MOLECULAR COMPONENTS OF THE CHANNELS UNDERLYING THE SUBTHRESHOLD - ACTIVATING A - TYPE K+ CURRENT.
AUTHOR: Nadal M S (Reprint); Ozaita A (Reprint); Vega Saenz de M E (Reprint); Amarillo Y (Reprint); Lau D (Reprint); Rudy B (Reprint)
AUTHOR ADDRESS: Dept Physiol and Neurosci, New York Univ School of Medicine, New York, NY, USA**USA
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner 2002 pAbstract No. 645.18 2002 2002
MEDIUM: cd-rom
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience Orlando, Florida, USA November 02-07, 2002; 20021102
SPONSOR: Society for Neuroscience

DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The somato-dendritic subthreshold-activating A-type K+ current (ISA) is a fast K+ current that activates transiently at membrane potentials that are below the threshold for Na+ spike generation, and plays important roles in neuronal excitability. Two key components of the channels underlying most of the ISA have been identified: Kv4 pore-forming subunits (mainly Kv4.2 and Kv4.3) and KChIP %%%associated%%% %%%proteins%%%. We have recently obtained evidence for the presence in rat brain mRNA of transcripts encoding a novel factor (termed KAF), which accelerates the kinetics of Kv4 channels, and could explain the fast kinetics of native ISA in many neurons.) Immunoaffinity purification was used to purify native Kv4 channel complexes solubilized from rat cerebellar membranes. Three specific and prominent bands are observed when the complex is dissociated and separated by SDS-PAGE. Immunoblotting and sequencing has shown that two of these bands correspond to KChIP and Kv4 polypeptides, respectively. A third polypeptide(s) of apprx115 kDa co-purifies specifically with the channel complex and shows an abundance comparable to that of Kv4 and KChIP polypeptides, suggesting it is an important component of native Kv4 channels. Purification and sequencing of this polypeptide will be carried out to explore whether it is responsible for KAF activity. In addition, we are using a functional cloning procedure, suppression cloning, to clone cDNAs encoding KAF.

8/7/6
DIALOG(R)File 5:Biosis Previews(R)

0013936169 BIOSIS NO.: 200200529680
Estradiol feedback alters potassium currents and firing properties of gonadotropin-releasing hormone neurons
AUTHOR: DeFazio R Anthony; Moenter Suzanne M (Reprint)
AUTHOR ADDRESS: Departments of Medicine and Cell Biology, University of Virginia, Jefferson Park Avenue, Room 7145 Multistory Building, P.O. Box 800578, Charlottesville, VA, 22908-0578, USA**USA
JOURNAL: Molecular Endocrinology 16 (10): p2255-2265 October, 2002 2002
MEDIUM: print
ISSN: 0888-8809
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: GnRH neurons are regulated by estradiol feedback through unknown mechanisms. Voltage-gated potassium channels determine the pattern of activity and response to synaptic inputs in many neurons. We used whole-cell patch-clamp to test whether estradiol feedback altered potassium currents in GnRH neurons. Adult mice were ovariectomized and some treated with estradiol implants to suppress reproductive neuroendocrine function; 1 wk later, brain slices were prepared for recording. Estradiol affected the amplitude, decay time, and the voltage dependence of both inactivation and activation of A-type potassium currents in these cells. Estradiol also altered a slowly inactivating current, IK. The estradiol-induced changes in IA contributed to marked changes in action potential properties. Estradiol increased excitability in GnRH neurons, decreasing both threshold and latency for action potential generation. To test whether estradiol altered phosphorylation of the channels or %%%associated%%% %%%proteins%%%, the broad-spectrum kinase inhibitor H7 was included in the recording pipette. H7 acutely reversed some but not all effects of estradiol on potassium currents. Estradiol did not affect IA or IK in paraventricular neurosecretory neurons, demonstrating a degree of specificity in these effects. Potassium channels are thus one target for estradiol regulation of GnRH neurons; this regulation involves changes in phosphorylation of %%%potassium%%% %%%channel%%% components.

8/7/7
DIALOG(R)File 5:Biosis Previews(R)

0013262405 BIOSIS NO.: 200100434244
Localization and enhanced current density of the Kv4.2 %%%potassium%%% %%%channel%%% by interaction with the actin-binding protein filamin
AUTHOR: Petrecca Kevin; Miller David M; Shrier Alvin (Reprint)
AUTHOR ADDRESS: Department of Physiology, McGill University, 3655 Drummond Street, Montreal, PQ, H3G 1Y6, Canada**Canada
JOURNAL: Journal of Neuroscience 20 (23): p8736-8744 December 1, 2000 2000
MEDIUM: print
ISSN: 0270-6474
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Kv4.2 potassium channels play a critical role in postsynaptic excitability. Immunocytochemical studies reveal a somatodendritic Kv4.2 expression pattern, with the channels concentrated mainly at dendritic spines. The molecular mechanism that underlies the localization of Kv4.2 to this subcellular region is unknown. We used the yeast two-hybrid system to identify the Kv4.2-%%%associated%%% %%%proteins%%% that are involved in channel localization. Here we demonstrate a direct interaction between Kv4.2 and the actin-binding protein, filamin. We show that Kv4.2 and filamin can be coimmunoprecipitated both in vitro and in brain and that Kv4.2 and filamin share an overlapping expression pattern in the cerebellum and cultured hippocampal neurons. To examine the functional consequences of this interaction, we expressed Kv4.2 in filamin+ and filamin- cells and performed immunocytochemical and electrophysiological analyses. Our results indicate that Kv4.2 colocalizes with filamin at filopodial roots in filamin+ cells but shows a nonspecific expression pattern in filamin- cells, with no localization to filopodial roots. Furthermore, the magnitude of whole-cell Kv4.2 current density is apprx2.7-fold larger in filamin+ cells as compared with these currents in filamin- cells. We propose that filamin may function as a scaffold protein in the postsynaptic density, mediating a direct link between Kv4.2 and the actin cytoskeleton, and that this interaction is essential for the generation of appropriate Kv4.2 current densities.

8/7/8
DIALOG(R)File 5:Biosis Previews(R)

0012954508 BIOSIS NO.: 200100126347
Interaction of large conductance, calcium-sensitive K+ channels (BKCa) with membrane-%%%associated%%% %%%proteins%%%
AUTHOR: Ling Shizhang (Reprint); Braun Janice (Reprint); Braun Andrew (Reprint)
AUTHOR ADDRESS: University of Calgary, Calgary, AB, Canada**Canada
JOURNAL: Biophysical Journal 80 (1 Part 2): p206a January, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the Biophysical Society Boston, Massachusetts, USA February 17-21, 2001; 20010217
SPONSOR: Biophysical Society
ISSN: 0006-3495
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

8/7/9
DIALOG(R)File 5:Biosis Previews(R)

0012936998 BIOSIS NO.: 200100108837
Generation and characterization of monoclonal antibodies against K and Na channel proteins
AUTHOR: Buchwalder L F (Reprint); Rhodes K J; Trimmer J S
AUTHOR ADDRESS: SUNY, Stony Brook, NY, USA**USA
JOURNAL: Society for Neuroscience Abstracts 26 (1-2): pAbstract No.-614.20

2000 2000
MEDIUM: print
CONFERENCE/MEETING: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000; 20001104
SPONSOR: Society for Neuroscience
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Generating antibodies against individual ion channel subunits is valuable for relating information from molecular cloning of channel genes to the contribution of the corresponding channel proteins in native neurons. Moreover, an immunological approach is often required due to the lack of natural or synthetic ligands that selectively bind many K and Na channel subtypes. While polyclonal antibodies (pAbs) can be useful for these purposes, mAbs offer many advantages over pAbs including high specificity, enhanced flexibility with subtype-specific detection methods, increased antibody yield and continuous supply. We developed novel strategies for producing and characterizing mAbs against alpha and beta subunit polypeptides of K and Na channels and %%%associated%%% %%%proteins%%%. First, mice are exposed to an antigen using a short, but intense immunization schedule. Then, hybridomas are produced and subjected to an extensive multistage screening strategy. The initial screen is a modified ELISA on transfected mammalian cells using chemiluminescence/luminometry. The mAbs selected from this screen are then assayed by immunofluorescence staining of mammalian cells transfected with subunit cDNAs. Where appropriate, cross-reactivity among related channel subtypes is examined using ELISA and immunofluorescence assays. Tertiary screens include immunoblotting of rat brain membranes and transfected cell lysates and immunohistochemical staining of nervous system tissues. Together, this approach has yielded a large collection of subtype-specific mAbs for neuronal K and Na channel subunits and %%%associated%%% %%%proteins%%%.

8/7/10
DIALOG(R)File 5:Biosis Previews(R)

0012894336 BIOSIS NO.: 200100066175
Modulation of Ca2+-activated potassium channels by cGMP-dependent signal transduction mechanism in cerebral arterial smooth muscle cells of the rabbit
AUTHOR: Han Jin; Kim Nari; Lee Kwangbok; Kim Euiyong (Reprint)
AUTHOR ADDRESS: Department of Physiology and Biophysics, College of Medicine, Inje University, Gaegeum-dong, Busanjin-gu, Busan, 614-735, South Korea**South Korea
JOURNAL: Korean Journal of Physiology and Pharmacology 4 (6): p445-453 December, 2000 2000
MEDIUM: print
ISSN: 1226-4512
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The present investigation tested the hypothesis that the activation of protein kinase G (PKG) leads to a phosphorylation of Ca2+-activated %%%potassium%%% %%%channel%%% (KCa channel) and is involved in the activation of KCa channel activity in cerebral arterial smooth muscle cells of the rabbit. Single-channel currents were recorded in cell-attached and inside-out patch configurations of patch-clamp techniques. Both molsidomine derivative 3-morpholinosydnonimine-N-ethylcarbamide (SIN-1, 50 muM) and 8-(4-Chlorophenylthio)-guanosine-3',5'-cyclic monophosphate (8-pCPT-cGMP, 100 muM), a membrane-permeable analogue of cGMP, increased the KCa channel activity in the cell-attached patch configuration, and the effect was removed upon washout of the drugs. In inside-out patches, single-channel current amplitude was not changed by SIN-1 and 8-pCPT-cGMP. Application of ATP (100 muM), cGMP (100 muM), ATP+cGMP (100 muM each), PKG (5 U/ mul), ATP (100 muM)+PKG (5 U/ mul), or cGMP (100

muM)+PKG (5 U/ mul) did not increase the channel activity. ATP (100 muM)+cGMP (100 muM)+PKG (5 U/ mul) added directly to the intracellular phase of inside-out patches increased the channel activity with no changes in the conductance. The heat-inactivated PKG had no effect on the channel activity, and the effect of PKG was inhibited by 8-(4-Chlorophenylthio)-guanosine-3',5'-cyclic monophosphate, Rp-isomer (Rp-pCPT-cGMP, 100 muM), a potent inhibitor of PKG or protein phosphatase 2A (PP2A, 1 U/ml). In the presence of okadaic acid (OA, 5 nM), PP2A had no effect on the channel activity. The KCa channel activity spontaneously decayed to the control level upon washout of ATP, cGMP and PKG, and this was prevented by OA (5 nM) in the medium. These results suggest that the PKG-mediated phosphorylations of KCa channels, or some %%%associated%%% %%%proteins%%% in the membrane patch increase the activity of the KCa channel, and the activation may be associated with the vasodilating action.

8/7/11
DIALOG(R)File 5:Biosis Previews(R)

0011679164 BIOSIS NO.: 199800473411
The atomic force microscope detects ATP-sensitive protein clusters in the plasma membrane of transformed MDCK cells
AUTHOR: Ehrenhoefer Uwe; Rakowska Agniewzka; Schneider Stefan Werner; Schwab Albrecht; Oberleithner Hans (Reprint)
AUTHOR ADDRESS: Dep. Physiol., Univ. Muenster, Robert-Koch-Str. 27a Innenhof, D-48149 Muenster, Germany**Germany
JOURNAL: Cell Biology International 21 (11): p737-746 Nov., 1997 1997
MEDIUM: print
ISSN: 1065-6995
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Plasma membrane proteins are supposed to form clusters that allow 'functional cross-talk' between individual molecules within nanometre distance. However, such hypothetical protein clusters have not yet been shown directly in native plasma membranes. Therefore, we developed a technique to get access to the inner face of the plasma membrane of cultured transformed kidney (MDCK) cells. The authors applied atomic force microscopy (AFM) to visualize clusters of native proteins protruding from the cytoplasmic membrane surface. We used the K+ channel blocker iberiotoxin (IBTX), a positively charged toxin molecule, that binds with high affinity to plasma membrane potassium channels and to atomically flat mica. Thus, apical plasma membranes could be 'glued' with IBTX to the mica surface with the cytosolic side of the membrane accessible to the scanning AFM tip. The topography of these native inside-out membrane patches was imaged with AFM in electrolyte solution mimicking the cytosol. The plasma membrane could be clearly identified as a lipid bilayer with the characteristic height of 4.9 +- 0.02 nm. Multiple proteins protruded from the lipid bilayer into the cytosolic space with molecule heights between 1 and 20 nm. Large protrusions were most likely protein clusters. Addition of the proteolytic enzyme pronase to the bath solution led to the disappearance of the proteins within minutes. The metabolic substrate ATP induced a shape-change of the protein clusters and smaller subunits became visible. ADP or the non-hydrolysable ATP analogue, ATP-gamma-S, could not exert similar effects. It is concluded that plasma membrane proteins (and/or membrane %%%associated%%% %%%proteins%%%) form 'functional clusters' in their native environment. The 'physiological' arrangement of the protein molecules within a cluster requires ATP.

8/7/12
DIALOG(R)File 5:Biosis Previews(R)

0011257138 BIOSIS NO.: 199800051385
Differential expression of isoforms of PSD-95 binding protein (GKAP/SAPAP1) during rat brain development

AUTHOR: Kawashima Nozomu; Takamiya Kogo; Sun Jie; Kitabatake Akira; Sobue Kenji (Reprint)
AUTHOR ADDRESS: Dep. Neurochem. Neuropharmacol., Biomed. Res. Cent., Osaka Univ. Med. Sch., 2-2 Yamadaoka, Suita, Osaka 565, Japan**Japan
JOURNAL: FEBS Letters 418 (3): p301-304 Dec. 1, 1997 1997
MEDIUM: print
ISSN: 0014-5793
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: PSD-95/SAP90, which binds to the C-terminus of NMDA receptor and Shaker-type %%%potassium%%% %%%channel%%%, is one of the major postsynaptic density proteins. Recently, novel classes of proteins interacting with the guanylate kinase domain of PSD95 have been identified, guanylate kinase-associated protein (GKAP) and SAP90/PSD-95-%%%associated%%% %%%proteins%%% (SAPAPs). Here we report the isolation of new isoforms of PSD-95 binding protein (GKAP/SAPAP1) using the yeast two-hybrid system. The isolated protein directly interacts with the guanylate kinase domain of PSD-95. Northern blot analyses revealed that the expression of these isoforms containing distinct N-terminal sequences is differentially regulated during brain development. The present findings suggest that each isoform of the PSD-95 binding protein is differentially expressed in a development-dependent manner and may be involved in the complex formation of PSD-95 and channel/receptors at the postsynaptic density.

8/7/13
DIALOG(R)File 5:Biosis Previews(R)

0010864781 BIOSIS NO.: 199799498841
Differential expression of the alpha and beta subunits of the large-conductance calcium-activated %%%potassium%%% %%%channel%%%: Implication for channel diversity
AUTHOR: Chang Chia-Ping (Reprint); Dworetzky Steven I; Wang Jianchen; Goldstein Margi E
AUTHOR ADDRESS: Central Nervous Syst. Drug Discovery, Bristol-Myers Squibb Pharm. Res. Inst., 5 Research Parkway, Wallingford, CT 06492, USA**USA
JOURNAL: Molecular Brain Research 45 (1): p33-40 1997 1997
ISSN: 0169-328X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In addition to the large a subunits that conduct selective ion currents, many native voltage-gated ion channels contain %%%associated%%% %%%proteins%%% which modulate the channel activity. Recently, a beta subunit of the large-conductance calcium-activated K+ (BK) channel has been cloned and functionally characterized. In this report, we studied the tissue distribution of the alpha and beta subunits of rat BK channels by nuclease protection analyses and in situ hybridization. BK-alpha mRNA is widely distributed but is especially enriched in the brain. In the adult brain, BK-alpha expression is robust and widespread throughout all areas of the neo-, olfactory and hippocampal cortices, habenula and cerebellum. Other prominent sites of BK-alpha expression include thalamus and amygdala. In marked contrast to the expression pattern of BK-alpha mRNA, the expression of BK-beta mRNA is relatively low and preferentially in the periphery. In rat brains, BK-beta mRNA occurs only in a few discrete populations of neurons that also express BK-alpha messages. These results indicate that the major type of BK channels in the brain, unlike the alpha-beta channel type in aortic and tracheal smooth muscle, is devoid of the beta subunit. These observations provide a structural basis for the BK channel diversity observed in a variety of tissues.

8/7/14
DIALOG(R)File 5:Biosis Previews(R)

0010694831 BIOSIS NO.: 199799328891
Protein kinase C stimulates the small-conductance K+ channel in the basolateral membrane of the CCD
AUTHOR: Lu Ming; Wang Wenhui (Reprint)
AUTHOR ADDRESS: Dep. Pharmacol., New York Med. Coll., Valhalla, NY 10595, USA**USA
JOURNAL: American Journal of Physiology 271 (5 PART 2): pF1045-F1051 1996 1996
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have used the patch-clamp technique to study the regulation of the activity of the basolateral small-conductance K+ channel (SK) in the cortical collecting duct (CCD) of the rat kidney. Addition of 50-75 nM calphostin C, an agent which specifically inhibits protein kinase C (PKC), reduced channel activity by 90% in cell-attached patches. In contrast, addition of 1 mu-M phorbol 12-myristate 13-acetate, a stimulator of PKC, led to addition of "new" K+ channel currents in 9 of 20 patches in the basolateral membrane of the CCD, and the mean increase in NP-o, a product of channel number (N) and open probability (P-o), was 0.90 in these 9 patches. However, application of 1 nM exogenous PKC had no significant effect on channel activity in inside-out patches, suggesting that the PKC effect on the activity of the SK observed in cell-attached patches was not a result of a membrane-delimited action, such as a direct phosphorylation of the SK or closely %%%associated%%% %%%proteins%%%. The effect of calphostin C on the SK can be reversed by addition of either 10 mu-M S-nitroso-N-acetylpenicillamine, a donor of nitric oxide, or 100 mu-M 8-bromoguanosine 3',5'-cyclic monophosphate. In addition, the inhibitory effect of calphostin C on the SK was completely abolished by pretreatment of the cells with 1 mu-M okadaic acid, an inhibitor of protein phosphatase. However, 100 mu-M N-omega-nitro-L-arginine methyl ester, an agent that inhibits nitric oxide synthases (NOS), blocked the SK in cell-attached patches in the presence of okadaic acid, suggesting that the effect of okadaic acid on calphostin C-induced inhibition of the SK was a step before formation of nitric oxide. We conclude that PKC is involved in the stimulation of the SK and that the effect of PKC on the SK may be mediated by regulation of NOS activity in the CCD of the rat kidney.

8/7/15
DIALOG(R)File 5:Biosis Previews(R)

0010184790 BIOSIS NO.: 199698652623
Immunological and physical characterization of the brain G protein-gated muscarinic %%%potassium%%% %%%channel%%%
AUTHOR: Inanobe Atsushi; Ito Hiroyuki; Ito Minoru; Hosoya Yukio; Kurachi Yoshihisa (Reprint)
AUTHOR ADDRESS: Dep. Pharmacol. II, Fac. Med., Osaka Univ., 2-2 Yamada-oka, Suita 565, Japan**Japan
JOURNAL: Biochemical and Biophysical Research Communications 217 (3): p 1238-1244 1995 1995
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The subunit composition of the G protein-gated inwardly rectifying K+ (K-G) channel protein extracted from mouse forebrain membranes was examined. A polyclonal antibody (anti-GIRK1C1) was prepared against the carboxyl terminal region of GIRK1, the major subunit of the K-G channel. The anti-GIRK1C1 IgG detected a single protein at apprx 65 kDa in SDS-PAGE of brain membranes. This IgG immunoprecipitated a macroprotein complex composed of GIRK1 and several %%%associated%%% %%%proteins%%% whose molecular weights ranging from 50 to 62 kDa on SDS-polyacrylamide gel. Upon size fractionation by both sucrose density gradient centrifugation and gel filtration, the solubilized K-G channel proteins migrated into a single peak, which suggests that the component

subunits form a single macromolecule. On the basis of the values of size fractionation, the molecular mass of the K-G channel macromolecule could be estimated at apprx 231,000. These results suggest that the K-G channel is most likely a tetrameric macroprotein composed of GIRK1 and co-immunoprecipitated proteins in the forebrain.

8/7/16
DIALOG(R)File 5:Biosis Previews(R)

0010090958 BIOSIS NO.: 199698558791
Development of the normal and hypertensive pulmonary vasculature
AUTHOR: Haworth Sheila G
AUTHOR ADDRESS: British Heart Found., Univ. London, 30 Guilford St., London WC1N 1EH, UK**UK
JOURNAL: Experimental Physiology 80 (5): p843-853 1995 1995
ISSN: 0958-0670
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Normal adaptation to extra-uterine life consists of an immediate increase in endothelial and smooth muscle cell (SMC) surface:volume ratio as the cells 'spread' in the vessel wall. Lumen diameter increases and resistance falls. Changes in SMC shape are associated with a transient depolymerization of contractile and cytoskeletal filaments. The four SMC phenotypes identified in the vessel wall rapidly show postnatal changes in the types of filament proteins and contractile-%%%associated%%% %%%proteins%%%, indicating that the term 'differentiation' means little at this age. At birth, all SMCs have a predominantly synthetic phenotype. Endothelium-dependent relaxation is relatively poor despite abundant nitric oxide synthase. SMCs are relatively insensitive to nitric oxide despite a high basal generation and a stimulated increase in cGMP generation. By contrast, the relaxation in response to ATP-sensitive potassium (K-ATP) channel activation is present at birth, the response being similar to that seen in the adult. Neonatal pulmonary hypertension, due to either congenital heart disease or experimental chronic hypobaric hypoxia (51 kPa) is associated with abnormal structural remodelling. In experimental pulmonary hypertension, the normal maturation of endothelium-dependent and -independent relaxation via soluble guanylate cyclase is delayed in newborns and the established responses are inhibited in older animals. The relaxant response to K-ATP channel activation is preserved. Thus, adaptation to extra-uterine life consists of a rapid sequence of integrated morphological and functional changes, which is disturbed by the presence of pulmonary hypertension. The pattern of recovery from a pulmonary hypertensive insult is determined by the age at exposure and type and duration of the insult.

8/7/17
DIALOG(R)File 5:Biosis Previews(R)

0008775698 BIOSIS NO.: 199395077964
The binding properties of the solubilized sulfonylurea receptor from a pancreatic B-cell line are modulated by the magnesium complex of ATP
AUTHOR: Schwanstecher Mathias (Reprint); Behrends Soenke; Brandt Christian; Panten Uwe
AUTHOR ADDRESS: Institute Pharmacology Toxicology, University Goettingen, Robert-Koch-Strasse 40, W-3400 Goettingen, Germany**Germany
JOURNAL: Journal of Pharmacology and Experimental Therapeutics 262 (2): p 495-502 1992
ISSN: 0022-3565
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Previous studies demonstrated that the Mg complex of ATP decreases glyburide- and increases diazoxide-binding to membranes from pancreatic islets. To examine further the mechanism of these effects, the

sulfonylurea receptors in microsomes of the hamster B-cell line HIT-T15 were solubilized with detergents. Maximum recovery of receptors (40%) was obtained with Triton X-100. Specific binding of (3H)glyburdie to the solubilized receptors (K-d = 0.35 nM, maximum number of binding sites = 170 fmol/mg of protein) corresponded well to specific binding to microsomes. In Triton X-100 extracts, MgATP (300 mu-M) reduced the number of high-affinity sites for (3H)glyburide by 50% and increased the dissociation constant for (3H)glyburide by 4-fold; MgATP was half-maximally effective at 20 mu-M. Development of MgATP-induced inhibition of (3H)glyburide binding to solubilized binding sites was not slower than dissociation of (3H)glyburide binding. Alkaline phosphatase accelerated the reversal of MgATP-induced inhibition of (3H)glyburide binding. In the presence of Mg++, not only ATP but also ADP, GTP and GDP inhibited (3H)glyburide binding to the solubilized receptor. However, MgADP did not inhibit (3H)glyburide binding when the MgATP concentration was kept low by the hexokinase reaction. MgATP significantly enhanced diazoxide-induced displacement of (3H)glyburide from the solubilized receptor. The MgATP-induced inhibition of binding was weakened by millimolar concentrations of free ATP. It is concluded that the binding sites for MgATP, glyburide and diazoxide are located at a single protein or at closely %%%associated%%% %%%proteins%%% which may include a protein kinase.

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15/3/1
DIALOG(R)File 5:Biosis Previews(R)

0013754567 BIOSIS NO.: 200200348078
Protein that enhances expression of potassium channels on cell surfaces and nucleic acids that encode the same
AUTHOR: %%%Brown Arthur M%%% (Reprint); Wible Barbara A; %%%Yang Qing%%%
AUTHOR ADDRESS: Brecksville, OH, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1258 (3): May 21, 2002 2002
MEDIUM: e-file
PATENT NUMBER: US 6391561 PATENT DATE GRANTED: May 21, 2002 20020521
PATENT CLASSIFICATION: 435-6 PATENT ASSIGNEE: The MetroHealth System
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

15/3/2
DIALOG(R)File 5:Biosis Previews(R)

0013268491 BIOSIS NO.: 200100440330
Protein that enhances expression of potassium channels on cell surfaces and nucleic acids that encode the same
AUTHOR: %%%Brown Arthur M%%% (Reprint); Wible Barbara A; %%%Yang Qing%%%
AUTHOR ADDRESS: Brecksville, OH, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1244 (4): Mar. 27, 2001 2001
MEDIUM: e-file
PATENT NUMBER: US 6207422 PATENT DATE GRANTED: March 27, 2001 20010327
PATENT CLASSIFICATION: 435-911 PATENT ASSIGNEE: The MetroHealth System, Cleveland, OH, USA PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

15/3/3
DIALOG(R)File 5:Biosis Previews(R)

0011472251 BIOSIS NO.: 199800266498
Cloning and expression of a novel K+ channel regulatory protein, KChAP
AUTHOR: Wible Barbara A (Reprint); %%%Yang Qing%%%; Kuryshev Yuri A; Accili Eric A; %%%Brown Arthur M%%%
AUTHOR ADDRESS: Rammelkamp Cent., 2500 MetroHealth Dr., Cleveland, OH 44109-1998, USA**USA
JOURNAL: Journal of Biological Chemistry 273 (19): p11745-11751 May 8, 1998 1998
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

15/3/4
DIALOG(R)File 5:Biosis Previews(R)

0011163379 BIOSIS NO.: 199799797439
Separable Kv-beta subunit domains alter expression and gating of potassium channels
AUTHOR: Accili Eric A; Kiehn Johann; %%%Yang Qing%%%; Wang Zhiguo; %%%Brown%%% %%% Arthur M%%%; Wible Barbara A (Reprint
AUTHOR ADDRESS: Rammelkamp Cent., 2500 MetroHealth Dr., Cleveland, OH 44109-1998, USA**USA
JOURNAL: Journal of Biological Chemistry 272 (41): p25824-25831 1997 1997
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English.

15/3/5
DIALOG(R)File 5:Biosis Previews(R)

0010667226 BIOSIS NO.: 199799301286
Single channel analysis of functional properties and mechanisms of K+ channel modulation by Kv-beta-1 subunits
AUTHOR: Wang Zhiguo (Reprint); Kiehn Johann; %%%Yang Qing%%%; Crumb William J; %%%Brown Arthur M%%%; Wible Barbara A
AUTHOR ADDRESS: Montreal Heart Inst., Montreal, PQ, Canada**Canada
JOURNAL: Circulation 94 (8 SUPPL.): pI5 1996 1996
CONFERENCE/MEETING: 69th Scientific Sessions of the American Heart Association New Orleans, Louisiana, USA November 10-13, 1996; 19961110
ISSN: 0009-7322
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

15/3/6
DIALOG(R)File 5:Biosis Previews(R)

0010653427 BIOSIS NO.: 199799287487
Comparison of binding and block produced by alternatively spliced Kv-beta-1 subunits
AUTHOR: Wang Zhiguo; Kiehn Johann; %%%Yang Qing%%%; %%%Brown Arthur M%%%; Wible Barbara A (Reprint
AUTHOR ADDRESS: Rammelkamp Cent. Res. MetroHealth Med. Cent., 2500 MetroHealth Dr., Cleveland, OH 44109-1998, USA**USA
JOURNAL: Journal of Biological Chemistry 271 (45): p28311-28317 1996 1996
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

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          $49.00  28 Types
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    $66.98  Estimated cost this search
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